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7.

ON ACUTE DILATATION OF THE HEART AS A CAUSE OF DEATH IN SCARLATINAL DROPSY.

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Some time ago I met with two cases of sudden or comparatively sudden death in the subjects of scarlatinal dropsy amongst my out-patients at the Evelina Hospital for Children, which were not to be explained satisfactorily by the modes of death which are most usual in that disease. Perhaps I ought to say the modes of death which are *thought* to be most usual, because I think that death in the manner which this paper records is not uncommon. I am not wrong in saying that death in scarlatinal dropsy is usually attributed to uræmic convulsions, to inflammation of the serous membranes of the thorax, or to œdema of the lungs, a part of the general anasarcous condition which is not inaptly termed "waterlogging." To these I would add acute dilatation of the left ventricle of the heart, which, I suspect, is not infrequent. The considerations which weigh with me in putting the following cases on record are several. First, that the possibility of such a termination is not sufficiently known. I do not say this of individual experience. Probably many are quite alive to the occurrence of sudden death from ventricular dilatation in acute nephritis, but it is not taught as one of the things generally

known, and individual experience is reduced to its minimum value so long as it wants the property of diffusibility. Secondly, till the condition is well recognised no treatment can be directed to avert its ill consequences; and thirdly, that to be forewarned of it is, in many cases, to be forearmed, whichever of two explanations we may accept of the phenomenon. The cases are as follows:

Case 1.—William P—, æt. 8, came to my out-patients' at the Evelina Hospital on June 27th. Scarlatina had been rife in the neighbourhood of his house, and about a fortnight before he came he had been languid and without appetite. No sore throat had been noticed, and the evidence of any pre-existing rash was very doubtful. His mother said, "No more than on his arm now," and this was a mottling of feeble capillary circulation. There were eight other children in the family and none of them had had scarlatina. Ten days before he came his face was noticed to be puffy, and he had since then been sick on and off. His urine had not been noticed except at nights, and then it was clear. He was peeling about the palmar surfaces of the hands and also about the elbows. His face was very puffy; the legs were œdematous and the thorax also.

On auscultation.—The lungs were normal, except that the resonance was not good towards their bases. The heart's action was forcible and quick, occasionally irregular, with a small, short, third beat. Tic tac, representing the normal first and second sound; occasionally there was a short interpolated tic-a-tac. The sounds were thick; the first long, not doubled. No marked increase of præcordial dulness. The position of the impulse is not recorded, so I presume it was normal. No urine was obtained at this visit. Some acetate of potash was ordered with some Liq. Ammoniæ Acetatis every three hours, and some compound jalap powder every morning. The mother was directed to keep the child perfectly at rest, and to feed him only upon fluid diet, of which he might partake freely.

I never saw the child again. He was taken worse on his way home, complaining much of pain in the epigastric region. He was sick and very restless, and died in this condition on June 22nd. He had no fits or loss of consciousness. The urine was very scanty.

The post-mortem was refused, and the nature of this case is therefore doubtful, but in the light of the others which follow, it seems to me not at all improbable that dilatation of the heart was the cause of the child's sudden death. The notes I made of the heart sounds are quite consistent with that probability, and the lungs did not at the time I saw him appear to be seriously affected. It is, however, possible that acute pleurisy, or peritonitis, or pericarditis may have supervened, and have been the cause of his pain and death.

Case 2.—Joseph J—, æt. 4, came to my out-patients' on January 31st, 1879. He had always been healthy till *six weeks* before his admission when he began to appear languid, and a lump came under the jaw, and he complained of his throat when washed. He never refused his food for pain when he swallowed. He had had no rash, and scarlatina was not known to be in the neighbourhood. *Three weeks* before he came his face began to swell. The child was brought to my colleague, Mr. Lucas, for the lump in its throat, and by him was transferred to me. The face and legs were very œdematous. There was much pallor. The heart's action was slow, eighty per minute. The sounds not thick. The impulse doubtfully external to the nipple. No irregularity. The chest was quite resonant, but râles could be heard over both lungs. The urine was smoky, and contained a good deal of albumen. I ordered Infus. Digitalis ʒss, Mist. Pot. Acet. ʒij every four hours, and twenty grains of Pulv. Jalapæ Co. every other morning.

The child did not appear to be particularly ill, and I did not apprehend any immediate ill consequences, and as the mother would not leave the child in the hospital it was taken home. Four days later it was too ill to be brought, and it died on the evening of the fourth day quite conscious, without any convulsions, but with very bad breathing.

I made an inspection forty-four hours after death. The body was well nourished. There was a good deal of anasarca of the legs and face and neck. The chest contained four or five ounces of serous fluid on both sides, and the lower lobe of the right lung had a little lymph upon it, and was in an early stage of pneumonia. On the other side the lung contained but little air, and was splenized in appearance. The pericardium was

healthy. The heart muscle was pale. The right ventricle full of clot of a pale watery character. The left side also distended with clots; its cavity considerably dilated and rounded; its muscular wall thin in proportion to the dilatation, but cutting rather toughly. The liver much congested. The kidneys were large, mottled, and smooth; their surfaces ecchymosed; the cortex swollen, confused, with abundance of fatty inflammatory products stuffed throughout it.

Case 3.—A girl, æt. 8, was admitted under the care of Dr. Pye-Smith in 1877. She had had measles, varicella, and whooping cough when much younger, but had enjoyed very good health till *five weeks* before her admission, when she woke up one night screaming violently, and on being taken out of bed to the light her mother noticed a red rash over her face and chest. She got better of this, but *fourteen days ago* her face and legs began to swell and the anasarca had gradually increased.

She was admitted in a dying condition, could only lie propped up in bed, breathing with great difficulty, and looking much distressed and emaciated. The legs and abdominal walls were œdematous. The urine was full of albumen, sp. gr. 1022. She was ordered some compound jalap powder, and Tr. Scillæ with Mist. Ammon. Acet. ℥ss 4tis horis, and four ounces of brandy. She died very soon after her admission.

I made the *post mortem* seventeen hours after death. There was much anasarca. The brain was healthy. The lower lobe of each lung was semi-solid from a state of œdema and splenisation combined. There was some, though not material, œdema glottidis. The pericardium was healthy. The *heart* weighed six ounces; its left ventricle was extremely dilated, so much so that at the apex very little muscle was left, and all the support derived from the interlacing of the muscular trabeculæ was done away with by their distension. There was even a little local bulging towards the exterior as if an aneurism of the heart were about to form. Some blood was extravasated into the muscular tissue at this part. Elsewhere the muscle was pale. The cavity contained two small, rather firm, decolorised ante-mortem coagula at the apex. The valves were healthy. On the right side there was ante-mortem coagulation in the right auricle with the formation of a cyst. The muscular

tissue of the right ventricle was thick and tough. The kidneys weighed six ounces; their surfaces were smooth, mottled with ecchymotic spots and numerous fat grains. Sections showed a swollen yellow fatty cortex, streaked here and there with congested vessels, and considerable lividity of the pyramids.

Case 4.—A boy, æt. 4½, was admitted into the Evelina Hospital, under the care of my colleague, Dr. Frederick Taylor, though it died before he saw it. The father stated that the boy had been ill about three weeks with headache and sickness. Some spots were noticed on the body at the first onset. He became better after a few days and continued so till *five days* before he was admitted, when he complained of sore throat, headache, and lumps in his neck. His face and eyelids then became puffy. His health had usually been good and there was no history of exposure. He had had but little sleep and was frequently retching. He was very pale and the body anasarcous; the feet and arms anasarcous. He was very restless, with dilating alæ nasi and orthopnœa. The chest was resonant. Pulse 143, small; temp. 101·8°; resp. 56. Urine smoky and full of albumen. He was ordered some compound jalap powder with excess of bitartrate of potash, which acted freely on the bowels; and some Liq. Potassæ Acetatis was given. The next day the pulse could hardly be felt. The respirations were 64 to 68. The temperature 99.° The bowels had acted freely. The bases of the lungs were dull. In this condition he died.

The *post-mortem* was made by Mr. Paley, and I was present. There was some amount of fluid in the pleural and peritoneal cavities, and also some increase of pericardial fluid. The lower lobe of each lung was partially collapsed, and there was but little air in any part of the left lung. The heart was enlarged, its left ventricle greatly dilated. The spleen was firm with an infarct near the surface. The kidneys enlarged and firm, the cortex pale in each (large white kidneys).

Case 5.—Kate W—, æt. 6, was admitted under the care of Dr. Moxon in 1876. I take the following notes from Mr. Sheldon's report:—

Two other children had died of scarlatina a fortnight before

her admission, and a month before she had herself been attacked with the same disease. She was on the point of getting up from this, apparently well, when her mother noticed a fortnight ago that her face was swollen. The dropsy increased rather rapidly, so she came to the hospital. She was a pale ill-looking child, with a great deal of swelling of the face and eyelids, and some general anasarca. The lips were dry, the tongue dry, and rather furred. She breathed with difficulty. Percussion over the chest was fair, except towards the bases, and the right base was less resonant than the left. Moist sounds were heard at the right base. Heart sounds faint, but normal; pulse 140. Urine passed, 29 ounces, slightly dusky: albumen, one fourth. It contained a large number of blood corpuscles and fragments of epithelial casts. Some compound jalap powder and acetate of ammonia mixture were ordered.

October 20th.—More œdema. The child is too ill to bear examination. The bowels have been freely open.

21st.—Respiration very short and quick, 60 per minute. Pulse 160. Temp. 102°. She expectorates a little thick tenacious mucus, with a streak of blood.

22nd.—Pulse hardly to be felt, but breathing more easily; temperature 101°. There is some diminished resonance all down the right lung behind. Expiration harsh and prolonged with moist râles. She became very restless in the afternoon, but died quietly.

I made the inspection twenty-two hours after death. Nothing worthy of mention was found except in the lungs, heart and kidneys, and of these I have the following notes:

Both lungs were in a state of extreme collapse, or rather in a state between simple collapse, and the solid state of pneumonia. They were neither so flaccid as in the one case, nor so solid as in the other; they were more like the lungs of mitral regurgitation. Probably collapse and œdema combined would explain the appearances. Hardly more than the apical third of each lung was unaffected. The *heart* weighed five ounces; the right side was over-distended. All the cavities were dilated, the left ventricle particularly so, and its walls not thick. *Kidneys.*—Rather swollen. Capsules normal. The cortex covered with numerous minute ecchymoses, and between these a yellow material speckled the surface. The section

showed a voluminous cortex, which was blurred and had lost its striæ. Under the microscope the epithelium was crowded and fatty in places, but on the whole by no means bad. The stroma had much interstitial small-cell growth throughout it, and it was thickly studded also with fat granules.

The notes I appended at the time of its occurrence to one of these cases will serve very well to epitomise the point of all, and to introduce the very few remarks that I wish to make upon them. "Great interest attaches to the fact that there was dilatation of the left ventricle of the heart. What was the cause of this? In the absence of any trace of valvular disease it is clear that it must have been due to some failure of the muscular tissue, either in doing ordinary work, or when extraordinary exertions were demanded of it. With regard to the former, the endocardium and pericardium, two great seats of disease so far as sources of paralysis of the cardiac muscle are concerned, were healthy, so that it only remains to suggest that the muscular tissue became spoiled during the scarlatinal pyrexia and yielded afterwards, or that the circumstances of acute Bright's disease are sufficient to induce bad nutrition of the muscular wall, and so to bring about the dilatation. On the other hand, there is good reason to suppose that the muscular wall yielded, not in consequence of the absolute failure of its strength and tone, but because extraordinary work was asked of it, to which it was unaccustomed." This is a very simple explanation; the tension in the arterial system becomes suddenly raised when there is acute inflammation of the kidney, the work proves too much for the left ventricle to perform, and the cavity becomes dilated. The main argument against this hypothesis is the one which experience would seem to inculcate, that death from dilatation of the heart is not usual in acute nephritis; but, at the same time, it is generally allowed that chronic Bright's disease is a frequent source of dilatation, and in most cases it is associated with hypertrophy also. So that it may be argued with much show of reason that if in chronic renal disease, where the heart has become in some measure accustomed to the strain, and hypertrophy has taken place, which must have added to the muscular power, dilatation has occurred notwithstanding; *à fortiori*, is it likely to occur when the strain comes suddenly upon a heart

in which there is no hypertrophy, and which is furnished only for habitual requisitions. Moreover, the clinical features of acute Bright's disease quite support this contention. That the tension is high I need not now stop to demonstrate, the sphygmograph has now proved it over and over again. But the heart itself gives evidence not unfrequently of considerable disturbance in its action, by irregularity, intermissions of the pulse, and occasional signs of increase of the size of the ventricle by displacement or other abnormality of the impulse. It is no improbable thing, therefore, to suppose that, now and then, perhaps when the depurative functions of the kidney are more than usually arrested, perhaps when the heart is in any way weak, perhaps under both sets of circumstances combined, a sudden dilatation of the left ventricle takes place, and death occurs by means of asystolism.

That is a short statement of the case: what follows is rather explanatory or commentative than a studied discussion. The facts do not admit of discussion. The only questions that allow of it are whether dilatation of the heart in acute nephritis is of frequent or of rare occurrence, and what are its causes when it does occur. Now, the first of these can only be answered by the sum of individual experiences, and at present, I say, we are without sufficient records of such cases. So I must content myself by pointing to these five cases, four of which leave no room for doubt; to the well-known disturbances of cardiac action which occur in acute as well as in other forms of nephritis; and to the results of observation by various sphygmographers, of which I may mention Dr. Mahomed's article "On the Pre-albuminuric Stage of Bright's Disease," in the 'Medico-Chirurgical Transactions,' which gives evidence of the suddenness of the increase of arterial tension; as the ground upon which I have come to form the opinion that acute dilatation of the heart is not so very uncommon. Secondly, what is the immediate cause, or what are the immediate causes of the dilatation? I have mentioned two conditions as competent, either singly or combined, to produce it. The altered composition of the blood in Bright's disease leads to obstruction in the peripheral circulation, and the heart has too much work to do. If this excess of work is but moderate the heart hypertrophies—Bright's original explanation of the hypertrophy of the heart;

which he found associated with renal disease—if it be more than can be accomplished the heart becomes dilated. It has been asserted by some that dilatation of the heart never occurs without hypertrophy, and this statement is mostly true if applied to chronic obstruction to the circulation. But it must not be taken, as is sometimes done, to disprove the occasional occurrence of cases such as are recorded here. Hypertrophy and dilatation combined are very common, and in a review of one's experience it would be quite correct to express a doubt whether, in chronic circulatory obstruction, dilatation is ever present unassociated with hypertrophy. But hypertrophy is the evidence of a chronic disease, and such a review would take no thought of acute cases which, though they may not be absolutely rare in practice, are certainly so on the post-mortem table. I have given above what appears to me to be a very strong argument in favour of acute dilatation over and above the actual demonstration of four cases, viz. that if dilatation occurs, as it confessedly does, in chronic disease, and when there is hypertrophy present to counteract it, much more is it likely to happen in acute disease, and when there is no such hypertrophy, provided that the circulation is similarly obstructed in both cases; and this has been shown to be the case. I had thought that this was a sufficient explanation of acute dilatation of the left ventricle, without taking any account of the question of the state of nutrition of the muscular wall, but a subsequent consideration of various points leads me now to think that the muscular wall itself may be more immediately at fault than I had at first imagined, and the point is one of importance with respect to the line of treatment to be adopted.

As a fact bearing upon this part of the question, I may quote the following case, which occurred in the post-mortem room at Guy's Hospital in January, 1878:

A girl, æt. 13, was admitted under the care of Dr. Moxon, on January 19th, 1878. It was stated that she had never been ill before. On December 29th she experienced soreness of the throat, and eleven days later came home from service with pains in her knees and other parts of her body, and subsequently her brother, nine years old, had scarlatina. Her wrists, ankles, and knees were red, swollen, and tender; her tongue dry, cracked, and glazed; and her fauces were still

inflamed. The heart's impulse was half an inch external to the nipple, and the præcordial dulness was increased. The first sound was roughened at the apex, and there was a systolic murmur at the base. The pulse was not hard. Urine, sp. gr. 1021, not albuminous at first; temp. 103°. She was afterwards noticed to be desquamating, albumen became abundant in her urine, she had much diarrhœa, and she died rather unexpectedly on the tenth day after her admission.

I made the inspection twenty-five hours after death. The liver, spleen, and tonsils were large, but a microscopic examination showed that the swelling was nothing more than acute turgescence due to congestion, at any rate it had in it nothing of a leukæmic nature.

The *heart* weighed 9 oz. The left ventricle was not dilated, indeed its cavity was rather small, but *the muscular tissue beneath its endocardium was distinctly striated from fatty degeneration, and the musculi papillares were fatty throughout.* The right auricle and ventricle were large and over-distended.

The *kidneys* weighed 13 oz., and were of a deep chocolate colour; the pelves full of a granular disintegrating blood clot. They were decomposing, but there could be no doubt that they were examples of extreme congestive nephritis.

Here is a case in which the muscular wall of the heart is found not dilated, but in a state of fatty degeneration after scarlatinal nephritis. Now, in this particular case, and in all others of *scarlatinal* dropsy, two causes are at work which may either of them lead to muscular degeneration, the scarlatina and the nephritis. Fever (pyrexia) is known to lead to granular changes in the muscles. I think it may also be said that granular change is very nearly related to fatty degeneration, and that it is possible for the one to pass into the other. So that, given the previous scarlatina, we find a state of things for which we are not altogether unprepared. But there is no post-mortem evidence to show that degeneration of the muscle is common in uncomplicated scarlatina, so that we shall not be wrong in attributing at any rate considerable importance to the nephritis. We tread upon much more certain ground here, because, first of all we have the common association of dilatation of the heart with chronic renal disease, to which I have already alluded; and secondly, Bright's disease is remarkable

for the *anæmia* which it produces. Now, there are several series of observations which show the profound importance of anæmia in determining malnutrition of the heart. Let me mention these.

The first is, that repeated hæmorrhage, enough to blanch the patient and cause a somewhat slow death, is occasionally followed by fatty degeneration of the heart. I have seen three such cases. The first was in an apparently healthy woman who had the misfortune to suffer from a uterine fibroid. She lost so much blood from this that one of her legs became gangrenous, and when she died, not long afterwards, there was very considerable fatty degeneration of the heart. The second was in a man, æt. 34, who died after long bleeding from a tumour of the bladder. The heart was fatty and dilated. The third was in a woman, æt. 23, under my own care last summer. She had had a miscarriage three months before, when she lost a large quantity of blood, and ever since, at intervals of five or six days she had been subject to fresh hæmorrhage. The left ventricle of the heart was a little dilated, and its muscular wall showed the tabby striation of advanced fatty degeneration.

2ndly. Fatty degeneration of the heart is almost constant in idiopathic anæmia, a disease of which nothing more is known except that it is a progressive and fatal anæmia which presents no other morbid changes.

3rdly. Fatty degeneration of the heart is not uncommon in leukæmia, which is an *anæmic* condition, and,

4thly. There is a large amount of clinical evidence to show that in women who suffer from chlorosis, or other persistent forms of anæmia, and in very many anæmic children, the heart's impulse is displaced, its action irregular, and the apex sounds thick or actually associated with murmurs; and these are signs which are best interpreted by the supposition that there is more or less temporary dilatation of the left ventricle.

These are the points which seem to me to render it not unlikely that dilatation of the heart in acute nephritis is due in part to muscular changes.

To sum up, then, the conclusions seem to be that acute dilatation of the heart is an occurrence to be prepared for and ready to treat in cases of acute nephritis, particularly after

scarlatina; and that two conditions probably take part in its production, (1) Sudden peripheral obstruction in the circulation, and (2), degenerative changes in the cardiac muscles. I must not, however, pass over the fact that the five cases which form the substance of this paper have all been children. One obvious reason of this is that they are all cases of scarlatinal dropsy, a disease *comparatively* uncommon in adult life. It may be said that this is an argument in favour of the febrile element being the more important one in the production of the condition, for acute nephritis is common enough in adults. To this, however, I should reply that acute nephritis in the same sense that scarlatinal nephritis is acute, is not common in adults. By far the greater number of cases of acute nephritis so-called have not anything like the sudden onset of scarlatinal nephritis, nor do they subside with the same rapidity; and it is quite evident that these two forms of disease are quite distinct. I must even go farther and say that many cases of acute tubal nephritis so-called have a creeping and insidious onset, and are only called acute because their first prominent symptom is the alarming one of dropsy, but this comes comparatively late in the disease. These cases are associated with *hypertrophy* as well as *dilatation*, and this is conclusive evidence upon the point.[1] But I see no reason to doubt that in the somewhat infrequent cases of acute nephritis in adults which are comparable to the cases we have cited as scarlatinal, there is the same risk of dilatation without hypertrophy to which I have called attention.

And now as to treatment. This is essentially a case where to be forewarned is to be forearmed, so that on this point I need add very little. We have to try to counteract or prevent the anæmia which is so constant in renal disease; to prevent if possible the arterial tension reaching so high a point as to endanger the functional integrity of the heart; or to relieve the

[1] There are a few cases on record which seem to show that hypertrophy of the heart may take place rapidly. I have lately had a boy under my own care whose heart weighed 19 ounces, and who stated that he had never been ill till three weeks before his admission. This statement was confirmed by the fact that the existing valvular disease was all recent ('Path. Soc. Trans.,' 1879). Other cases have been collected by Dr. Stone in the Croonian Lectures at the Royal College of Physicians for 1879. But these are few in number, and are, I think, no more than the exceptions which prove the rule as stated in the text.

tension if it should occur. For the first I think something may be done by the free and persistent use of the milder forms of iron, and both for the first and the second indications much may be done by the timely administration of the usual eliminants, the hot air bath, sudorifics, and particularly purgatives. Of the respective value of these remedies—I would say, that there are no drugs which act so speedily as well as powerfully on arterial tension as hydragogue cathartics, but I would emphasise the word *timely* in the foregoing sentence, because it is one thing to give purgatives to *prevent* high tension, and quite another to give them for the reduction of tension in the late stages of nephritis when the kidney is in such a state of disease as to render them practically useless. In either case they may do good, but in the latter they suffer in reputation by the defeat which they eventually undergo. In the former they are often successful. I once asked a medical man of very large experience what he did in his cases of scarlatinal dropsy. His reply was that he never had any, "because," said he, "I always take care to keep the bowels well open by means of castor oil during the period of convalescence." I have no doubt my friend is not singular in his experience of the great value of such a treatment, and it is a good illustration of timely or preventive medication. In those cases where we have no opportunities to be beforehand with the disease, and where it is the pressing need of the hour to reduce the strain which is thrown upon the heart, I still think the remedies to be trusted are chiefly purgatives, but I am also distinctly of opinion that cardiac tonics, particularly the infusion of digitalis, are not to be neglected. The use of such drugs as these is at first sight unscientific, it seems parallel to the administration of ergot for the expulsion of the fœtus when there is unyielding obstruction of the passage in front. But the two cases are not parallel. It is well known that digitalis acts not alone upon the heart, but that it aids the circulation by its action upon the muscular coat of the arterioles. Moreover, there is, as between the heart and the arteries, a certain amount of accommodation, and there is always plenty of storage capacity in the vessels which may be made to relieve the heart if it can be utilised in time. This may be done sometimes by temporarily stimulating the heart to act more

vigorously, and sometimes by paralysing the peripheral vessels, by nitrite of amyl and other like remedies. To this it must be added that experience teaches that, though much is known concerning the behaviour of digitalis, our knowledge is not yet so complete that it can be expressed in definite formulæ; and it is a fact that the action of this drug is sometimes most beneficial in cases where it has been prescribed beforehand with misgiving and hesitation. So that, allowing the case did not argue well for the administration of the drug, it would, nevertheless, not be right to withhold it if the danger were urgent, and no other remedy offered like *chances*. Digitalis is a drug which, in failure of the left ventricle from over-work, is always given with some risk, but often rescues the patient from speedy death; and there is no remedy given at such times which is free from risk.

And here I should conclude, but that a case occurred to me last year so closely akin to these that it is well worth associating with them.

I was asked to make a *post-mortem* examination upon a gentleman who had died quite unexpectedly. He was a barrister, æt. 62. He had suffered from some trifling ailment for a few days before, consisting chiefly of sharp lumbago, but he had been sufficiently well the night before his death to be up till late, and he expressed his intention of going to business again on the morrow. His relatives had noticed, however, that he had not been well lately, and he had *always* been subject to attacks of pallor, during which he would appear to be quite ill and then quickly regain his strength. He woke his family early on the morning of his death, complained of shivering, and said he felt as if he were going to die. His usual medical attendant was summoned, but death occurred before he arrived. There was some doubtful gouty history and he once had a bad quinsy. His habits had always been strictly temperate.

The notes of the inspection are headed—*Syncope, Old Apoplexy, Congested Kidneys, Thick Vessels.*

Head.—The vessels at the base of the brain were thickened. The brain externally looked quite normal, and no disease was found anywhere except a small old brown softening from bygone apoplexy on the left side. This was half an inch in extent, and situated outside the lenticular nucleus of the corpus striatum, but

just breaking into it near the middle of its outer border. The pons, medulla, and ventricles were all quite healthy. There was a little œdema of the ary-epiglottic folds, but not sufficient to narrow the rima. A little less development of the right crico-arytænoideus posticus than the left was noted, but no existing cause for this could be found. The diaphragm was healthy. The *heart* was about normal in weight. *Its cavities all contained black post-mortem clot, and were all flaccid.* The muscular tissue was good, and the walls not thickened. The valves of the heart were healthy. The coronary arteries were thick. The *kidneys* were of full size, weighing about 12 ounces; Very dark in colour, indurated, and containing one or two cysts. Their structure looked healthy, and this was subsequently confirmed by microscopical examination. The vessels were decidedly thick.

It will be unnecessary to go on with the descriptions of negative results that are appended to the various viscera. It will be enough to take the note I made at the end of the report. "The only definite cause of death that could be suggested was syncope dependent upon high vascular tension. This hypothesis seems consonant with the old apoplexy, the thickened vessels, and the flaccid heart. The absence of hypertrophy is not altogether against this view, as I have met with other cases more obviously syncopal than this in renal disease where the heart was not enlarged; and it seems that in proportion to the acuteness of the onset of high tension is the risk of sudden syncope." The question of angina pectoris was also considered, as the state of the coronary arteries suggested the possibility of its occurrence, but the history gave no indication whatever in that direction.

This case was one where

> "Life struck sharp on death
> Makes awful lightning."

The friends of the patient were quite unprepared for anything of the kind, as he had been possessed of uniformly good health. It would have been very much more satisfactory to have met, adopting the ugly phraseology of the present day (more to protest against it than from necessity), with some "coarse" disease the account of which would have carried conviction to the minds of his relatives. But what did they know about high tension

and syncope? It was impossible to explain it to them, and had it been possible it would not have been wise, for it carried with it an exaggeratedly unpleasant reflection for an unprofessional mind, that it was an end from which apparent health gave no immunity, and that any one else, the most hale looking amongst them, might drop suddenly in the same way. That, indeed, is partly the wonder of the doctor, not that such cases occur, but that they do not present themselves more often.

The case finished, so far as I was concerned, in a very suggestive way, for happening to meet the son of this gentleman some time afterwards, I did attempt to explain to him in some measure this dependence of syncope on high tension, and he closed the conversation with this remark: "But that is very strange, for my father always had such a remarkably strong pulse." "A remarkably strong pulse," methinks, would hardly obtain so favorable a verdict in a court with the sphygmograph as the presiding judge.

The case was at the time more especially interesting to me as illustrating the difficulty that there is sometimes, and not very infrequently either, of putting one's finger upon the precise cause of death. If the interpretation of this case is the right one, here is a cause of sudden death which may leave very little evidence behind it.

P.S.—I have two omissions to record:—1st. My colleague, Mr. Howse, tells me that some years ago Sir William Gull was in the habit of giving special prominence to the fact that acute pericarditis in rheumatic fever is liable to give rise to rapid dilatation of the heart, which is often mistaken for pericardial effusion. Sir W. Gull also taught that dilatation is succeeded, not preceded, by hypertrophy in some of these cases, and Mr. Howse has used this as an illustration in discussing the conditions associated with varices.[1] I am very glad to be able to mention this, not only because it is a weighty confirmation of some of the remarks made in this paper from another point of view; but also because it has hitherto been unrecorded. And yet as an observation of one whose clinical experience is of the widest, upon a still disputed point in cardiac pathology, it is exactly the kind of record which is wanted.

2nd. Concerning the rate at which the heart hypertrophies, I have accidentally omitted to refer to Dr. Peacock's original observations which were published in the 'Edinburgh Monthly Journal,' vol. vii, p. 405, and are again alluded to in his 'Croonian Lectures' for 1865.

[1] 'Guy's Hospital Reports,' 1877, vol. xxii, p. 481.